U.S. Department of Labor

Office of Administrative Law Judges
525 Vine Street - Suite 900
Cincinnati, Ohio 45202

(513) 684-3252
(513) 684-6108 (FAX)

Issue date: 28Sep2001

Case No.: 1998-BLA-0336
BRB No.: 99-761 BLA

In the Matter of

JOE HURT
Claimant

v.

LOCUST GROVE COAL COMPANY
Employer

KENTUCKY COAL PRODUCERS SELF-INSURANCE FUND
Carrier

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

DECISION AND ORDER ON REMAND - AWARDING BENEFITS

On March 30, 1999, I issued a Decision and Order - Dismissing Henderson Branch and Awarding Benefits in the above-captioned case. I found that Locust Grove was the responsible operator for this claim, that the claimant established a material change in condition pursuant to § 725.309(d) in that he was now totally disabled from a pulmonary or respiratory standpoint, that the medical opinion evidence established pneumoconiosis, and that the medical opinions also established that the claimant's total disability was due to pneumoconiosis. The employer appealed that decision to the Benefits Review Board ("the Board"). On September 29, 2000, the Board issued its Decision and Order affirming in part, vacating in part, and remanding the claim for further consideration consistent with its opinion. The findings as to responsible operator, material change in condition, and total disability were unchallenged on appeal and thus affirmed. The Board found error with the weighing of the evidence as to pneumoconiosis and causation of total disability, and remanded the case for further consideration of those issues.

After consideration of the parties' arguments on the amendments to the regulations, I issued an Order on June 5, 2001 allowing adjudication of this claim to proceed. I found that the amended regulations will not affect the outcome of the case as the changed subsections which could impact the case are mere codifications of existing case law or no evidence has been submitted which would trigger that particular amended regulation. (June 5, 2001 Order).

Pneumoconiosis

Dr. Fino's opinion

Dr. Fino cited several reasons for concluding that the claimant did not have "an occupationally acquired pulmonary condition as a result of coal mine dust exposure." They were:

1. The majority of chest x-ray readings are negative for pneumoconiosis.

2. The spirometric evaluations that have been performed show an obstructive ventilatory abnormality based on the reduction in the FEV1/FVC ratio. This obstructive ventilatory abnormality has occurred in the absence of any interstitial abnormality. In addition, the obstruction shows involvement in the small airways. Large airway flow is measured by the FEV1 and FEV1/FVC ratio. Small airway flow is measured by the FEF 25-75. On a proportional basis, the small airway is more reduced than the large airway flow. This type of finding is not consistent with a coal dust related condition but is consistent with conditions such as cigarette smoking, pulmonary emphysema, non-occupational chronic bronchitis, and asthma. Minimal obstructive lung disease has been described in working coal miners and has been called industrial bronchitis. This condition is characterized by cough and mucous production plus minimal decreases in the FEV1 in some miners. Industrial bronchitis resolves within six months of leaving the mines. Obstructive lung disease may also arise from coal workers' pneumoconiosis when significant fibrosis is present. The fibrosis results in the obstruction. In this case, although obstruction can be seen in coal workers' pneumoconiosis, the obstruction is unrelated to coal mine dust exposure.

3. There is improvement following bronchodilators on the pulmonary function studies. Reversibility following bronchodilators implies that the cause of the obstruction is not fixed and permanent. Certainly, pneumoconiosis is a fixed condition. Because it is fixed, bronchodilator medication would be of no benefit. One cannot improve on an abnormality caused by coal workers' pneumoconiosis. Hence, improvement following bronchodilators showing reversibility to the overall pulmonary impairment is clearly evidence of a non-occupationally acquired pulmonary condition causing the obstruction.

4. Lung volumes are used to "look" at the consistency of lung tissue. **. . .** [T]hey are a measure of whether the lung is of normal consistency, whether it is over-inflated, or whether it is under-inflated. Over-inflated conditions are due to obstructive lung disease. Under-inflated conditions are due to contraction due to fibrotic scarring as is seen in pulmonary fibrosis. This patient has elevated lung volumes. There is stale air trapped in his lungs due to his obstructive lung disease. This is a typical pattern that we see in individuals who have obstructive lung diseases such as emphysema, or asthma, or chronic obstructive bronchitis, or any combination of the three. This is not a pattern consistent with the contraction of lung tissue due to fibrosis as would be expected in simple coal workers' pneumoconiosis.

5. Mild resting hypoxia varies over time. For instance, there was no hypoxia on 1/26/93; however, there was hypoxia on 8/2/93. The hypoxia then went away by 11/2/93. Variability in hypoxia or for that matter any type of lung abnormality is not consistent with a coal mine dust related pulmonary condition. Coal mine dust related pulmonary conditions are permanent. As such, there would not be variability over time. Variability in hypoxia over time is consistent with asthma which is the diagnosis that I would reach in this case.

6. There is no improvement in oxygen transfer.

(EXLG 2).

As to Dr. Fino's opinion, I found that:

Although Dr. Fino's opinion is slightly different from Dr. Broudy's, in that he stated that a disabling *obstructive* impairment is associated only with complicated pneumoconiosis, I find his opinion to be as hostile-to-the-Act as Dr. Broudy's. The newly submitted evidence shows that the claimant has a mixed impairment, both restrictive and obstructive, although the obstruction is greater than the restriction and the condition is referred to as chronic *obstructive* pulmonary disease. **...** Dr. Fino implicitly considered both kinds of pulmonary impairment in reaching his conclusion that only complicated pneumoconiosis could cause the changes seen in the claimant. Yet the Regulations provide that a claimant may be found to be totally disabled due to simple pneumoconiosis based on qualifying pulmonary function study results. §§ 718.202(a), 718.204(c)(1). Additionally, I find that Dr. Fino's opinion shows a bias against the claimant. He did not address the evidence favorable to the claimant. As a reviewing physician, he had the opportunity to review all of the medical evidence,

> but in justifying his conclusion, he addressed only the bits and pieces favorable to it. *See Underwood v. Elkay Mining, Inc.*, 105 F.3d 946, 951 (4th Cir. 1997) (to the extent that the administrative law judges determine that a particular expert's opinion is not, in fact, independently based on the facts of a particular claim, but is instead influenced more by the identity of his or her employer, the administrative law judges have clear discretion to disregard such an expert's opinion as being of exceedingly low probative value). The claimant's condition has not been shown to be totally reversible. The FEV1 is still qualifying after bronchodilators. Dr. Fino's argument on reversibility and variability also does not take into account that two conditions may exist at the same time, and provided no reasoning for why he feels that the effects of coal dust are an "all or nothing" proposition. For these reasons, I give Dr. Fino's opinion no weight.

(March 30, 1999 Decision and Order pp. 21-22).

The Board found that:

> [I]t is unclear how the administrative law judge also found Dr. Fino's opinion deficient. The administrative law judge apparently equated complicated pneumoconiosis with the "significant fibrosis" Dr. Fino alluded to, which is a mischaracterization of Dr. Fino's statement. Moreover, in finding that Dr. Fino did not address the evidence favorable to claimant, it is not clear whether the administrative law judge is simply disagreeing with Dr. Fino's ultimate conclusion or there is evidence favorable to claimant which Dr. Fino failed to address and the omission of the specific evidence has been found to constitute bias. If the administrative law judge is finding the latter, there needs to be some discussion of that evidence.

(September 29, 2000 Decision and Order at p. 5).

As to the latter, I specified that I found Dr. Fino's opinion to be biased because "[t]he claimant's condition has not been shown to be totally reversible. The FEV1 is still qualifying after bronchodilators. Dr. Fino's argument on reversibility and variability also does not take into account that two conditions may exist at the same time, and provided no reasoning for why he feels that the effects of coal dust are an all or nothing proposition." I focused on the factors of reversibility, the decrease in the FEV1, and variability because they were factors cited by Dr. Fino in relating the impairment to asthma alone.

The claimant was born on December 31, 1938, and thus was fifty-four years old at the time of the pulmonary function studies on January 26, 1993 and August 3, 1993. (DX 1, 9, 11). The tables at Appendix B to Part 718 indicate that a fifty-four year old man's FEV1 at 60% of predicted is

2.16 at 70.1 inches and 2.19 at 70.5 inches. The two studies in question respectively show post-bronchodilator FEV1s of 1.3 and 2.04, still disabling values despite the administration of a bronchodilator. As such, at least the 40% decrease in predicted FEV1 which resulted in the claimant meeting the disability criteria, is not reversible. This substantial irreversibility was not addressed by Dr. Fino, even though he also stated that "pneumoconiosis is a fixed condition. Because it is fixed, bronchodilator medication would be of no benefit." Dr. Fino provided no basis for ruling out pneumoconiosis as a cause of any of that irreversible decrease in the FEV1.

Dr. Fino did state that industrial bronchitis might result "in minimal decreases in the FEV1 in some miners" and that "[o]bstructive lung disease may also arise from coal workers' pneumoconiosis when significant fibrosis is present." Yet this is counter to the Act. Compare his opinion to the Regulations. Dr. Fino stated that it is the reduction in the FEV1/FVC ratio and the FEV1 that defines obstruction and a decrease in the large airway flow, the same reductions that the Regulations look to to determine total disability due to pneumoconiosis. 20 C.F.R. § 718.204(c)(1). The Regulations thus provide that decreases in the FEV1 and FEV1/FVC ratio support a claimant's claim for benefits under the Act, whether the claim is simple medical pneumoconiosis or legal pneumoconiosis.[1] The Regulations do not set forth that these changes will only be seen with "significant fibrosis." They are used to determine total disability even if the readings are 1/0 or negative.

Dr. Fino stated that the obstruction was also not due to a coal dust related condition because the reduction in the FEF 25-75 (small airway flow) was proportionally greater than the reduction in the FEV1 and FEV1/FVC ratio (large airway flow). This is a point not addressed by any other physician, the Regulations, or the comments to the Regulations. His argument may have merit, but it is overshadowed by his requirement of fibrosis. Despite his tailored wording of "an occupationally acquired pulmonary condition as a result of coal mine dust exposure," and notwithstanding his statement on industrial bronchitis (a condition he believes to be temporary), Dr. Fino is clearly stuck on medical pneumoconiosis. Paragraph one concerns x-ray evidence of pulmonary fibrosis. Paragraph 2 addresses obstruction due to fibrosis. Paragraph 4 specifies that he looked for "a pattern consistent with the contraction of lung tissue due to fibrosis as would be expected in simple coal workers' pneumoconiosis." In *Cornett v. Benham Coal, Inc.*, 227 F.3d 569 (2000), the United States Court of Appeals for the Sixth Circuit found that:

> Dr. Fino attempted to explain his rationale for completely excluding Cornett's exposure to coal dust as an aggravating factor. Dr. Fino attributed Cornett's obstructive lung disease solely to cigarette smoking because, in his opinion, the pulmonary function tests were not consistent with "fibrosis as would be expected in simple coal workers' pneumoconiosis."

---

[1] There is an irrebutable presumption of total disability with complicated pneumoconiosis. 20 C.F.R. § 718.304.

The court held that "although 'fibrosis' is generally associated with 'medical' pneumoconiosis, it is not a required element of the broader concept of 'legal' pneumoconiosis. **...** The legal definition does not require 'fibrosis' but instead requires evidence that coal dust exposure aggravated the respiratory condition." In keeping with this precedent, I have reviewed Dr. Fino's opinion and found that it does not address the issue of legal pneumoconiosis, but only medical pneumoconiosis.

For these reasons, I again accord no weight to the opinion of Dr. Fino. His opinion is not well-reasoned, is hostile-to-the-Act, and does not address the broader category of legal pneumoconiosis.

Dr. Broudy's opinion

The Board affirmed my finding that Dr. Broudy's opinion was hostile-to-the-Act, based on his belief that disabling impairments can be caused only by complicated pneumoconiosis, not simple.

I also note that at his deposition, Dr. Broudy made clear that his opinion was limited to medical pneumoconiosis as he defined coal workers' pneumoconiosis as:

> [A] disease of the lungs which results from the deposition of coal dust and related nonorganic materials into the lung and the tissue reaction to that deposition in the lungs. It is manifested on X-ray usually with nodular or sometimes linear opacities which are usually symmetrically distributed throughout the lung zones.
>
> . . . .
>
> **...** [T]he subject would, of course, have to have a history of adequate exposure, which varies depending on the intensity and duration of exposure. And, furthermore, he would need to have either a chest X-ray which is suggestive of pneumoconiosis with reasonable exclusion of other possible causes of the X-ray findings, or he could have biopsy or even autopsy evidence of pneumoconiosis.

(DX 60, depo. at pp. 8-9).

Interestingly, contrary to Dr. Fino, Dr. Broudy testified that "the ventilatory studies are not generally diagnostic tools, especially in this type of determination, but are used to determine the patient's respiratory capacity." (DX 60, depo. at pp. 10-11). His conclusion that the obstruction was not due to pneumoconiosis was based on his belief that simple pneumoconiosis does not cause a significant or substantial decrease in pulmonary function (DX 60, depo. at p. 25), again a belief that the Board upheld as being hostile-to-the-Act.

Dr. Harrison's opinion

Dr. Harrison examined the claimant in relation to his state claim for benefits. Patient history included that the claimant "gets up and uses a Proventil inhaler with relief. He has been on Theo-Dur in the past but this led to abdominal difficulties and therefore he doesn't take it anymore." Medical history was "asthma"; neither COPD nor any other pulmonary condition was mentioned. Dr. Harrison concluded that:

> Mr. Hurt has a negative reading for pneumoconiosis under the ILO classification. He does, however, have significant obstructive lung disease which is under estimated by the FEV-1/FVC ratio because he has such severe air trapping(residual volume of 216). I don't feel that at the present time he has thepulmonary capacity to do the work of a miner or arduous manual labor. This is,however, due to his underlying asthma rather than an occupational exposure. He is on therapy with inhaled beta agonists, Azmacort for his asthma, but it is not adequate and he needs more intensive care. I would also recommend follow up of the right hilum **...** .

The form Dr. Harrison used indicates that a miner is to be diagnosed with an occupational lung disease caused by his coal mine employment only if the x-ray reading is 1/0 or greater, which is apparently the state law. (DX 14).

As with the opinions of Drs. Fino and Broudy, I find that Dr. Harrison did not properly address the possibility of pneumoconiosis as defined under the federal regulations. While he indicated that he considered occupational exposure as a cause of the obstruction, Dr. Harrison apparently ruled out pneumoconiosis because of the negative x-ray, which is contrary to the Act. He provided no explanation for not relating the claimant's obstruction to coal dust exposure. He simply stated that it was related to asthma, a condition he believed the claimant was diagnosed with and treated for. His use of the word "underlying" also implies that he may believe there is a condition superimposed on the asthma, which he did not address. I therefore find that Dr. Harrison's opinion is entitled to little weight.

Dr. Myers' opinion

Like Dr. Harrison, Dr. Myers filled out a state form in regards to his examination of the claimant. Patient history included that:

> Presently he carries Nitroglycerin with him for his chest pain. He uses a Proventil inhaler for his lungs. He takes Proventil drops and Zestril and Verapamil for his high blood pressure and heart trouble. **...** He has been hospitalized for blood poisoning and his heart problem. He fractured a toe in the 70's.

Dr. Myers diagnosed "perennial asthma, progressive, Class III and meeting federal Black Lung criteria under Regulation 718, Appendix B; and arteriosclerotic hypertensive cardiovascular disease with angina with effort, Class III under AMA Guidelines." He related the changes on the pulmonary function study and the claimant's total disability to both diagnoses. (DX 13).

While Dr. Myers' opinion makes some reference to the federal regulations, it nevertheless does not address the statutory definition of pneumoconiosis. Therefore, it is not known if he considered and why he ruled out coal dust exposure as a cause of the obstruction or as an aggravating factor of the asthma. Accordingly, I also give his opinion little weight.

The opinions of Drs. Baker and Wicker

The Board vacated my finding on the opinions of Drs. Wicker and Baker for failure to "explain what specific factors the physicians relied upon, other than x-rays and a history of

exposure, in concluding that the claimant's impairment was due to coal mine employment or how the opinions of Drs. Wicker and Baker were well-reasoned and documented."

Dr. Baker's opinions show that, in addition to a positive x-ray for Category 1 pneumoconiosis and the occupational history, he related the Claimant's impairment to coal dust exposure and cigarette smoking due to the pulmonary function study finding of a severe obstructive ventilatory defect with a mild to moderate degree of restriction. (DX 64). He testified that the claimant had a sufficient number of years of exposure to coal dust [at least ten] to contract the disease of coal workers' pneumoconiosis, and that while the claimant did not have fifteen pack-years of smoking, he nevertheless would attribute some of the obstruction to smoking due to the combination of smoking and coal dust exposure creating a worse effect that either one acting by itself. He concluded that the claimant did not have asthma, but did have an element of bronchial spasm. (DX 64, depo. at pp. 4-5, 10, 13-14). He explained that in order for him to diagnose coal workers' pneumoconiosis, he would need to find present:

> [A] history of dust exposure of ten years or more and take into account individual susceptibility and the type of job. Secondly, optimally x-ray changes. And, last, any type of chronic pulmonary disorder in association with dust exposure which would be

> one criteria for the diagnosis of coal workers' pneumoconiosis, any type of chronic respiratory impairment associated with coal dust exposure.

(DX 64, depo. at p. 12).

Dr. Wicker also considered an x-ray showing of Category 1 pneumoconiosis and the occupational history, as well as an abnormal pulmonary function study and physical examination finding of a few expiratory wheezes. Dr. Wicker stated that "[t]he patient has a history of cigarette smoking [12 pack years, ending in 1972] and it is felt that his disability arises both from his cigarette smoking as well as his exposure to coal dust." (DX 15).

Again, I find these opinions to be documented and well-reasoned. They recognize that both coal dust exposure and cigarette smoking can cause obstructive lung disease, and that because the claimant has a sufficient exposure to both, the obstruction is attributable to both. I find no flaw in that reasoning. In *Cornett*, *supra*, where the court was presented with the opinions of Drs. Vaezy and Baker that "the obstructive ventilatory defect could have been caused by either smoking or coal dust exposure," it pointed out that "[a]lthough neither report eliminated smoking as a cause, both doctors were unequivocal that coal dust exposure aggravated Cornett's pulmonary problems, thus supporting the existence of 'legal,' although possibly not 'medical' pneumoconiosis." The court further stated that "accurately following the regulatory definition of pneumoconiosis cannot be grounds for rejecting a doctor's opinion."

The employer continues to argue that Dr. Wicker's opinion that the claimant has pneumoconiosis is not credible because he did not continually list a diagnosis of pneumoconiosis on each examination. For the reasons previously given, I again find this argument to be without merit. Additionally, I note that Dr. Wicker also did not continually list obesity as a diagnosis despite the claimant's weight being essentially the same, nor did he list chronic low back pain every time.

The employer argues that Dr. Wicker did not diagnose legal pneumoconiosis, only medical. However, COPD is a broad category including emphysema, chronic bronchitis, and asthma, which if related to coal dust exposure,[2] equates to legal pneumoconiosis, 20 C.F.R. § 718.201, thereby making

---

[2] The comments to the new Part 718 regulations state that:

> The term "chronic obstructive pulmonary disease" (COPD) includes three disease processes characterized by airway dysfunction: chronic bronchitis, emphysema and asthma. Airflow limitations and shortness of breath are features of COPD, and lung function testing is

the terms somewhat interchangable. Dr. Wicker made findings of "pneumoconiosis." He made findings of "chronic bronchitis." He also made findings of "COPD." In his letter of September 24, 1993, Dr. Wicker stated that "[m]y diagnosis of pneumoconiosis rests on the basis of both his chest x-ray and his COPD accounts for the vast majority of his changes on PFTs." (DX 29). I interpreted this sentence as stating that he diagnosed pneumoconiosis based on both the positive x-ray reading and the obstruction, accounting for the vast majority of the changes on the pulmonary function studies. He could not have meant to say that he was basing his diagnosis of pneumoconiosis on two chest x-rays, since he had only reviewed one x-ray (dated August 2, 1993) up to that time. My interpretation is consistent with the August 3, 1993 examination report in which Dr. Wicker unquestionably stated that the claimant's pulmonary impairment was due to both cigarette smoking and coal dust exposure. (DX 15). I therefore reaffirm my previous finding that Dr. Wicker diagnosed legal pneumoconiosis in addition to medical pneumoconiosis.

The diagnosis of asthma

The Board found that I did not explain "why the diagnosis of asthma by Drs. Myers, Broudy, Fino and Branscomb was a misdiagnosis." The Board then went on to state that I had interpreted the medical tests and substituted my conclusions for them. (September 29, 2000 Decision and Order at pp. 5-6).

As to Dr. Branscomb's opinion, I found that:

> His summary that the claimant has been treated for asthma since the 1980s is not supported by the records he reviewed. Rather, Dr. Branscomb is assuming that the treatment was for asthma because he concluded that the claimant has asthma. The

---

> used to establish its presence. Clinical studies, pathological findings, and scientific evidence regarding the cellular mechanisms of lung injury link, in a substantial way, coal mine dust exposure to pulmonary impairment and chronic obstructive lung disease. **...** NIOSH concluded that "[i]n addition to the risk of simple CWP and PMF [progressive massive fibrosis], epidemiological studies have shown that coal miners have an increased risk of developing COPD." **...**
>
> Drs. Fino and Bahl disagree, but the Department believes that their opinions are not in accord with the prevailing view of the medical community or the substantial weight of the medical and scientific literature. **...**
>
> 65 Fed. Reg. 79939 (December 20, 2000).

> reports of Dr. Wicker, the claimant's treating physician, do not show a diagnosis of asthma. Although Drs. Myers, Harrison, and Broudy diagnosed asthma, none of them are treating physicians, and as such, did not prescribe the claimant's medications. Contrary to Dr. Branscomb's summary of Dr. Broudy's deposition, Dr. Broudy did not state that the claimant was begun on medication for asthma and bronchitis in 1982. The history Dr. Broudy obtained was that the claimant was begun on medication for bronchitis in 1982. Dr. Broudy opined that the medication was appropriate for chronic obstructive pulmonary disease or bronchial asthma. (DX 60 p. 17). I further note that the previously submitted records show diagnoses of pneumoconiosis, chronic bronchitis, emphysema, and chronic obstructive pulmonary disease, but do not show any diagnosis of asthma. Further, those records do not show any history of asthma for the claimant nor his family. (DX 56). Thus, asthma is a relatively new consideration and not the given that Dr. Branscomb presumes it to be. The tone of Dr. Branscomb's report is thus inappropriate, for it is unreasonable to expect the claimant to "admit" to having asthma when no treating physician has diagnosed him with it. For these reasons, I give Dr. Branscomb's opinion very little probative weight.

Considering the tone of a report is like considering the demeanor of a witness who testifies. In his report, Dr. Branscomb stated that "[a]s was true with a number of other examinations, Mr. Hurt denied any history of asthma." (EXLG 4 at p. 2). In each of the summaries of those examinations, Dr. Branscomb stated that claimant "denied" having asthma. With this wording and repetitiveness, Dr. Branscomb implied that the claimant knew he had asthma and was lying about it. But as already set forth, the medical records do not show any diagnosis of asthma by a treating physician. They do show a diagnosis of bronchitis, as the claimant has stated he has. Thus, it was unreasonable for Dr. Branscomb to expect the claimant to "admit" to having asthma.

Dr. Branscomb also presented a diagnosis of asthma as a given because of the medications used. But the evidence shows that the medications prescribed are not prescribed for asthmatics alone, and Dr. Branscomb did not address this. His opinion does not show any recognition of Dr. Wicker as the treating physician, or any inquiry as to who prescribed the medications and as to why. Dr. Wicker's records show that he prescribed the medications of Proventil, Proventil inhaler, and Azmacort, and that he did not diagnose asthma. (CX 1).

Dr. Branscomb is entitled to reach his own diagnosis. However, he is a reviewing physician, and his diagnosis of asthma must be weighed in that context. Dr. Wicker, the treating physician, never indicated a diagnosis of asthma. Whether or not he would agree that a component of the COPD is asthma (apparently no one thought to ask him), Dr. Wicker has nevertheless related the claimant's impairment/COPD to coal dust exposure and cigarette smoking.

As to why he did not diagnose asthma, Dr. Baker testified that:

> [W]e didn't do bronchial dilator studies and I don't have any history of reversibility. His history is mostly that of a consistent daily difficulty with his breathing. People who have asthma have good periods and bad periods. He's had significant difficulty on an every day and every night basis for a period of time of eight to ten years. I thin (sic) he has bronchial spasm which can mimic asthma and with the symptoms that he has, but with the persistent nature of daily symptoms over a long period of time with no history of improvement and otherwise wouldn't show [an FEV1] improvement following administration of bronchial dilators of greater than 20 percent.

(DX 64, depo. at p. 14). However, at no point, did Dr. Baker state that a finding of asthma would change his opinion on causality. The employer's summation of his testimony was "that asthma, if it existed, could play a part." (DX 64, depo. at p. 11).

I note that the January 26, 1993 pulmonary function study did not show any improvement post-bronchodilator (DX 9), but that the August 3, 1993 study did (DX 11). On the latter study, the FEV1 went from 36% of predicted to 57%.

I again give greatest weight to the opinion of the treating physician, Dr. Wicker. The last pulmonary function study was in 1993. Since then, the claimant's treatment has been based on his complaints to his doctor, and the doctor's observations and examinations of him. As gathered from other medical opinions in this record, reversibility is a major factor upon which a diagnosis of asthma is based. The physician who prescribed the medications and is in the best position to know what type of relief the claimant is getting from day-to-day, is Dr. Wicker. He did not diagnose asthma. Instead, he diagnosed pneumoconiosis, chronic bronchitis, and COPD, with changes on the pulmonary function study due to cigarette smoking and coal dust exposure.

Causation of Total Disability

Drs. Baker and Wicker opined that the claimant's totally disabling pulmonary condition is due to cigarette smoking and coal dust exposure. I already found that their opinions are documented and well-reasoned.

Drs. Myers and Harrison related the claimant's impairment in part or in total to asthma, without addressing the broad definition of pneumoconiosis at § 718.201 and § 718.202(a)(4) (which permits a diagnosis of pneumoconiosis notwithstanding a negative x-ray), whether coal dust could have caused that type of COPD, and whether coal dust could have worsened a pre-existing condition of asthma. I therefore find that their opinions are not probative on this issue.

Dr. Broudy's opinion was upheld as hostile-to-the-Act.

I give Dr. Fino's opinion no weight for the reasons already stated.

Dr. Branscomb concluded that the claimant was totally disabled due to asthma. Again, I find his diagnosis of asthma outweighed by the opinion of Dr. Wicker, the treating physician.

For these reasons, I reaffirm my previous finding that the claimant's total disability is due to pneumoconiosis.

Attorney's Fee

Claimant's counsel has thirty days to submit an application for an attorney's fee. The application shall be prepared in strict accordance with 20 C.F.R. §§ 725.365 and 725.366. The application must be served on all parties, including the claimant, and proof of service must be filed with the application. The parties are allowed thirty days following service of the application to file objections to the fee application.

ORDER

IT IS THEREFORE ORDERED THAT the March 30, 1999 Decision and Order - Dismissing Henderson Branch and Awarding Benefits is AFFIRMED. Locust Grove Coal Company is ORDERED to pay benefits in accordance with that Decision.

A
JOSEPH E. KANE
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 CFR § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order by filing a Notice of Appeal with the Benefits Review Board at Post Office Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.